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FILE COVERS 1969 TO DATE.

CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT

FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 5 December 2001 (20011205/ED)

The BIOSIS file has been reloaded. Enter HELP RLOAD and HELP REINDEXING for details.

=> s angiogenesis

L1 14823 ANGIOGENESIS

=> s inhibitor?

L2 510716 INHIBITOR?

=> s l1 and l2

L3 2772 L1 AND L2

=> s plasminogen

L4 29697 PLASMINOGEN

=> s l3 and l4

L5 270 L3 AND L4

=> s (tumor? or tumour?)

609110 TUMOR?

52663 TUMOUR?

L6 628523 (TUMOR? OR TUMOUR?)

=> s l5 and l6

L7 149 L5 AND L6

=> s endothelial

L8 100270 ENDOTHELIAL

=> s l7 and l6

L9 149 L7 AND L6

=> s l7 and l8

L10 77 L7 AND L8

=> s peptide?

L11 264452 PEPTIDE?

=> s l10 and l11

L12 4 L10 AND L11

=> d l12 1-4 ab bib

L12 ANSWER 1 OF 4 BIOSIS COPYRIGHT 2001 BIOSIS

AB The urokinase **plasminogen** activator system is involved in **angiogenesis** and **tumor** growth of malignant gliomas, which are highly neovascularized and so may be amenable to antiangiogenic therapy. In this paper, we describe the activity of A6, an octamer capped **peptide** derived from the non-receptor-binding region of urokinase **plasminogen** activator. A6 inhibited human microvascular **endothelial** cell migration but had no effect on the proliferation of human microvascular **endothelial** cells or U87MG glioma cells in vitro. In contrast, A6 or cisplatin (CDDP) alone suppressed subcutaneous **tumor** growth in viva by 48% and 53%, respectively, and, more strikingly, the combination of A6 plus CDDP inhibited **tumor** growth by 92%. Such combination treatment also greatly reduced the volume of intracranial **tumor** xenografts and increased survival of **tumor**-bearing animals when compared with CDDP or A6 alone. **Tumors** from the combination treatment group had significantly reduced neovascularization, suggesting a mechanism involving A6-mediated inhibition of **endothelial** cell motility, thereby eliciting vascular sensitivity to CDDP-mediated toxicity. These data suggest that the combination of an **angiogenesis inhibitor** that targets **endothelial** cells with a cytotoxic agent may be a useful therapeutic approach.

AN 2000:434985 BIOSIS

DN PREV200000434985

TI A **peptide** derived from the non-receptor-binding region of urokinase **plasminogen** activator inhibits glioblastoma growth and **angiogenesis** in vivo in combination with cisplatin.

AU Mishima, Kazuhiko; Mazar, Andrew P.; Gown, Allen; Skelly, Marilyn; Ji, Xiang-Dong; Wang, Xu-Dong; Jones, Terence R.; Cavenee, Webster K.; Huang, H.-J. Su (1)

CS (1) Ludwig Institute for Cancer Research, San Diego Branch, 9500 Gilman Drive, La Jolla, CA, 92093-0660 USA

SO Proceedings of the National Academy of Sciences of the United States of America, (July 18, 2000) Vol. 97, No. 15, pp. 8484-8489. print. ISSN: 0027-8424.

DT Article

LA English

SL English

L12 ANSWER 2 OF 4 BIOSIS COPYRIGHT 2001 BIOSIS

AB Angiostatin, a potent **inhibitor** of **angiogenesis**, **tumour** growth and metastasis, is a biologically active fragment of **plasminogen**, containing the kringle domains 1-4. It is generated from **plasminogen** by limited proteolysis. We show that prostate-specific antigen (PSA), a serine proteinase secreted by human prostate and human prostate cancer cells, is able to convert Lys-**plasminogen** to biologically active angiostatin-like fragments, containing kringles 1-4, by limited proteolysis of **peptide** bond Glu439-Ala440 in vitro. In an in vitro morphogenesis assay, the purified angiostatin-like fragments inhibited proliferation and tubular formation of human umbilical vein **endothelial** cells with the same efficacy as angiostatin. This finding might help to understand growth characteristics of prostate cancer, which usually has low microvessel

density and slow proliferation.

AN 2000:48872 BIOSIS

DN PREV200000048872

TI Generation of angiostatin-like fragments from **plasminogen** by prostate-specific antigen.

AU Heidtmann, H.-H. (1); Nettelbeck, D. M.; Mingels, A.; Jaeger, R.; Welker, H.-G.; Kontermann, R. E.

CS (1) St Joseph Hospital, Wiener Strasse 1, D-27568, Bremerhaven Germany

SO British Journal of Cancer, (Dec., 1999) Vol. 81, No. 8, pp. 1269-1273. ISSN: 0007-0920.

DT Article

LA English

SL English

L12 ANSWER 3 OF 4 BIOSIS COPYRIGHT 2001 BIOSIS

AB Angiostatin is a potent **inhibitor** of **tumor angiogenesis** and the growth of metastatic foci. Recent studies have indicated that neoplastic cells can generate angiostatin directly or in cooperation with **tumor**-associated macrophages. In studies reported here, we determined whether angiostatin is generated in mice under non-neoplastic settings. Utilizing murine RAW264.7 macrophages and thioglycollate-elicited peritoneal macrophages, we demonstrate that angiostatin-like fragments are generated as a byproduct of the

proteolytic

regulation of membrane-bound plasmin. Plasmin proteolysis and subsequent loss in membrane-bound plasmin activity requires active plasmin but was unaffected by **inhibitors** of metalloproteinases. Lysine binding fragments of plasmin, isolated from macrophage-conditioned media

utilizing

affinity chromatography, appeared as a major (48 kDa) and two minor bands (42 and 50 kDa) in SDS-polyacrylamide gel electrophoresis and were immunoreactive with anti-kringle 1-3 IgG. Each **peptide** begins with Lys77 and contains the entire sequence of angiostatin. The affinity isolated plasma fragments inhibited bFGF-induced **endothelial** cell proliferation. Lavage fluid recovered from the peritoneal cavities

of

mice previously injected with thioglycollate contained angiostatin-like plasmin fragments similar to those generated in vitro. This is the first demonstration that angiostatin-like plasmin fragments are generated in a non-neoplastic inflammatory setting. Thus, in addition to regulating pericellular plasmin activity, proteolysis of plasmin generates inactive kringle-containing fragments expressing angiostatic properties.

AN 1999:12824 BIOSIS

DN PREV199900012824

TI Macrophage formation of angiostatin during inflammation. A byproduct of the activation of **plasminogen**.

AU Falcone, Domenick J. (1); Khan, K. M. Faisal; Layne, Tiffany; Fernandes, Lianne

CS (1) Dep. Pathol., Rm. A678, Cornell Univ. Med. Coll., 1300 York Ave., New York, NY 10021 USA

SO Journal of Biological Chemistry, (Nov. 20, 1998) Vol. 273, No. 47, pp. 31480-31485.

ISSN: 0021-9258.

DT Article

LA English

L12 ANSWER 4 OF 4 BIOSIS COPYRIGHT 2001 BIOSIS

AB At present the most used method to quantify **tumor angiogenesis** in human solid **tumors** is the count of intratumoral microvessels in the primary lesion. This method requires the use of specific markers to vascular endothelium and of immunohistochemical

procedures to visualize microvessels. Several studies have found that intratumoral microvessel density (IMD) determined in the primary **tumor** is significantly associated with metastasis and prognosis in

some solid neoplasia, particularly in operable breast carcinoma. The subjective evaluation of IMD made by two observers at the microscope is rapid and of low cost, but presents some difficulties, mainly the identification of the most vascularized area ("hot-spot") within each tumor. This method can be improved upon to attain a better reproducibility among different pathologists. For example, the use of a multiparametric computerized image analysis system (CIAS) seems to be a promising tool to improve accuracy, feasibility and reproducibility of microvessel counts, although there are still some open technical problems to completely automate its use. Angiogenic activity is the result of a balance between angiogenic stimuli and angio-inhibition. Therefore the determination of angiogenic peptides and/or natural angiogenesis inhibitors in the tumor tissue, serum, or urine of cancer patients seems to be a promising alternative to microvessel counting. At present it is possible to determine the expression of basic fibroblast growth factor (bFGF), vascular endothelial growth factor, and transforming growth factor beta using immunohistochemical methods. Serum and urine levels of bFGF can be assessed using an immunoenzymatic assay. Methods used to assess the expression and levels of urokinase-type plasminogen activator (uPA) or plasminogen activator inhibitor-1 (PAI-1) have also been developed, and correlate with angiogenic activity and prognosis of patients with breast cancer. Finally, some investigational methods to assess angiogenesis in vivo are presented and discussed. Angiogenesis is a very complex phenomenon. Thus it seems reasonable to hypothesize that its assessment by using concurrently several of the available methods may provide more valid, accurate, and comprehensive information on the angiogenic activity of each single tumor. For a reliable and reproducible assessment of angiogenesis for all of the assays, validation procedures and quality control protocols are mandatory.

AN 1995:456490 BIOSIS
 DN PREV199598470790
 TI Novel methods for the determination of the angiogenic activity of human tumors.
 AU Barbareschi, Mattia; Gasparini, Giampietro (1); Morelli, Laca; Forti, Stefano; Dalla Palma, Paolo
 CS (1) Dep. Oncol., St. Sortolo Med. Cent., 36100 Vicenza Italy
 SO Breast Cancer Research and Treatment, (1995) Vol. 36, No. 2, pp. 181-192.
 ISSN: 0167-6806.
 DT General Review
 LA English

=> file uspatfull

COST IN U.S. DOLLARS	SINCE FILE	TOTAL
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FULL ESTIMATED COST	12.04	12.19

FILE 'USPATFULL' ENTERED AT 17:48:30 ON 08 DEC 2001
 CA INDEXING COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY (ACS)

FILE COVERS 1971 TO PATENT PUBLICATION DATE: 6 Dec 2001 (20011206/PD)
 FILE LAST UPDATED: 6 Dec 2001 (20011206/ED)
 HIGHEST GRANTED PATENT NUMBER: US6249914
 HIGHEST APPLICATION PUBLICATION NUMBER: US2001049836
 CA INDEXING IS CURRENT THROUGH 6 Dec 2001 (20011206/UPCA)
 ISSUE CLASS FIELDS (/INCL) CURRENT THROUGH: 6 Dec 2001 (20011206/PD)
 REVISED CLASS FIELDS (/NCL) LAST RELOADED: Apr 2001
 USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Apr 2001

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>>> (IPC) Manuals, editions 1-6, in the /IC1, /IC2, /IC3, /IC4, <<<
>>> /IC5, and /IC (/IC6) fields, respectively. The thesauri in <<<
>>> the /IC5 and /IC fields include the corresponding catchword <<<
>>> terms from the IPC subject headings and subheadings. <<<

This file contains CAS Registry Numbers for easy and accurate
substance identification.

=> s 112

3101 ANGIOGENESIS
118591 INHIBITOR?
5750 PLASMINOGEN
40757 TUMOR?
3998 TUMOUR?
11592 ENDOTHELIAL
53756 PEPTIDE?
L13 258 L10 AND L11

=> s adenocarcinoma

L14 4351 ADENOCARCINOMA

=> s 113 and 114

L15 68 L13 AND L14

=> d 115 1-10 ab bib

L15 ANSWER 1 OF 68 USPATFULL

AB Although ribonucleases are characterized by the hydrolysis of RNA,
these enzymes perform many functions, including anti-parasitic activity,
anti-bacterial activity, and anti-viral activity. Ribonucleases are
also

known to possess anti-neoplastic activity, and **angiogenesis**
-stimulating activity. "Zrnase1" is a new member of the human
ribonuclease family.
AN 2001:224217 USPATFULL
TI Human ribonuclease
IN Conklin, Darrell C., Seattle, WA, United States
PI US 2001049434 A1 20011206
AI US 2001-801231 A1 20010307 (9)
PRAI US 2000-187917 20000308 (60)
DT Utility
FS APPLICATION
LREP Phillip Jones, ZymoGenetics, Inc., 1201 Eastlake Avenue East, Seattle,
WA, 98102
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 3760

L15 ANSWER 2 OF 68 USPATFULL

AB The present invention relates to polynucleotide and polypeptide

molecules for zFGF12 a novel member of the FGF family. The present invention also includes antibodies to the zFGF polypeptides, and methods of using the polynucleotides and polypeptides.

AN 2001:212528 USPATFULL
TI Novel FGF Homolog zFGF12
IN Conklin, Darrell C., Seattle, WA, United States
PI US 2001044525 A1 20011122
AI US 2001-754634 A1 20010104 (9)
PRAI US 2000-174582 20000105 (60)
DT Utility
FS APPLICATION
LREP Deborah A. Sawislak, ZymoGenetics, Inc., 1201 Eastlake Avenue East, Seattle, WA, 98102
CLMN Number of Claims: 18
ECL Exemplary Claim: 1
DRWN 8 Drawing Page(s)
LN.CNT 2457

L15 ANSWER 3 OF 68 USPATFULL

AB The present invention relates to compositions and methods for preventing

the development of epithelial ovarian cancer by administering compounds in an amount capable of regulating TGF- β expression in the ovarian epithelium and/or capable of optimally altering expression of other surrogate biomarkers identified by microarray technology. HRT and OCP regimens comprising such compositions and methods are disclosed.

AN 2001:212435 USPATFULL
TI Prevention of ovarian cancer by administration of products that induce biologic effects in the ovarian epithelium
IN Rodriguez, Gustavo C., Durham, NC, United States
PI US 2001044431 A1 20011122
AI US 2001-798453 A1 20010302 (9)
RLI Continuation-in-part of Ser. No. US 2000-528963, filed on 21 Mar 2000, PENDING
DT Utility
FS APPLICATION
LREP Raymond N. Nimrod, Suite 1000, 200 South Michigan Avenue, Chicago, IL, 60604
CLMN Number of Claims: 33
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 4240

L15 ANSWER 4 OF 68 USPATFULL

AB The invention relates to method for identifying prostatic intraepithelial neoplasia, methods for determining metastatic potential of **tumors**, and to methods and compositions for inhibiting or preventing metastasis of cancers. In one aspect, the invention provides a method to determine metastatic potential of **tumors**, particularly prostatic **tumors**. In another aspect, the invention provides a method of identifying prostate cancer associated conditions, particularly prostatic intraepithelial neoplasia. In these regards, the invention relate to determining protein or mRNA of effectors of arachidonic acid release, particularly uteroglobin protein or mRNA, to identify intermediate conditions such as PIN or to gauge metastatic potential of prostatic **tumors**.

The invention also relates to methods and compositions that prevent or inhibit metastasis of cancers. In this regard, the invention particularly relates to methods and compositions that inhibit arachidonic acid, those that inhibit phospholipase A₂. More particularly in this regard, the invention relates to uteroglobin or muteins, **peptide** analogs or mimetics of uteroglobin and lipocortins or muteins, **peptide** analogs, or mimetics of lipocortins that inhibit metastasis. Especially it relates to methods

and compositions in which uteroglobins, particularly human
uteroglobins,
inhibit or prevent metastasis of cancer, particularly prostatic cancer.
AN 2001:202599 USPATFULL
TI Uteroglobin therapy for epithelial cell cancer
IN Patierno, Steven R., 2906 Brook Dr., Falls Church, VA, United States
22042
Manyak, Michael J., 2322 Blaine Dr., Chevy Chase, MD, United States
20815
PI US 6316416 B1 20011113
AI US 2000-512385 20000225 (9)
RLI Division of Ser. No. US 1997-966196, filed on 7 Nov 1997, now patented,
Pat. No. US 6054320 Division of Ser. No. US 1996-658796, filed on 5 Jun
1996, now patented, Pat. No. US 5935860, issued on 10 Aug 1999
Continuation-in-part of Ser. No. US 1995-486203, filed on 7 Jun 1995,
now patented, Pat. No. US 5830640, issued on 3 Nov 1998
Continuation-in-part of Ser. No. US 1995-400084, filed on 7 Mar 1995,
now patented, Pat. No. US 5696092, issued on 9 Dec 1997
DT Utility
FS GRANTED
EXNAM Primary Examiner: Seaman, D. Margaret
CLMN Number of Claims: 89
ECL Exemplary Claim: 1
DRWN 5 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 2488
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L15 ANSWER 5 OF 68 USPATFULL

AB A method for treating and alleviating melanomas and various cancers
characterized by the expression of VEGF-D by the **tumor**, the
method comprising screening to find an organism with **tumor**
cells expressing VEGF-D and administering an effective amount of a
VEGF-D antagonist to prevent binding of VEGF-D; methods for screening
for neoplastic diseases, where detection of VEGF-D on or in cells such
as **tumor** cells, blood vessel **endothelial** cells,
lymph vessel **endothelial** cells, and/or cells with potential
neoplastic growth indicates neoplastic disease; a method for promoting
and maintaining vascularization of normal tissue in an organism by
administering VEGF-D or a fragment or analog thereof; methods for
screening **tumors** for metastatic risk where expression of
VEGF-D by the **tumor** indicates metastatic risk; and methods to
detect micro-metastasis of neoplastic disease where detection of VEGF-D
on or in a tissue sample indicates metastasis of neoplastic disease.

AN 2001:199742 USPATFULL

TI Methods for treating various cancers expressing vascular
endothelial growth factor D, for screening for a neoplastic
disease and for maintaining vascularization of tissue

IN Achen, Marc, Victoria, Australia
Stacker, Steven, Victoria, Australia

PI US 2001038842 A1 20011108

AI US 2001-796714 A1 20010302 (9)

PRAI US 2000-186361 20000302 (60)

DT Utility

FS APPLICATION

LREP CROWELL & MORING LLP, Intellectual Property Group, P. O. Box 14300,
Washington, DC, 20044-4300

CLMN Number of Claims: 44

ECL Exemplary Claim: 1

DRWN 15 Drawing Page(s)

LN.CNT 1724

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L15 ANSWER 6 OF 68 USPATFULL

AB Disclosed is the surprising discovery that aminophospholipids, such as
phosphatidylserine and phosphatidylethanolamine, are specific,

accessible and stable markers of the luminal surface of **tumor** blood vessels. The present invention thus provides aminophospholipid-targeted diagnostic and therapeutic constructs for use in **tumor** intervention. Antibody-therapeutic agent conjugates and constructs that bind to aminophospholipids are particularly provided, as are methods of specifically delivering therapeutic agents, including toxins and coagulants, to the stably-expressed aminophospholipids of **tumor** blood vessels, thereby inducing thrombosis, necrosis and **tumor** regression.

AN 2001:196603 USPATFULL
TI Cancer treatment methods using therapeutic conjugates that bind to aminophospholipids
IN Thorpe, Philip E., Dallas, TX, United States
Ran, Sophia, Dallas, TX, United States
PA Board of Regents, The University of Texas System, Austin, TX, United States (U.S. corporation)
PI US 6312694 B1 20011106
AI US 1999-351457 19990712 (9)
PRAI US 1998-92589 19980713 (60)
US 1998-110600 19981202 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Bansal, Geetha P.
LREP Williams, Morgan & Amerson
CLMN Number of Claims: 50
ECL Exemplary Claim: 1,2,3,4
DRWN 6 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 8243
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L15 ANSWER 7 OF 68 USPATFULL

AB The present invention relates to novel transcription control elements, including promoters, derived from **angiogenesis**-related genes, particularly the mouse VEGF gene, the mouse VEGFR-2 receptor gene, and the mouse Tie2 gene. Also disclosed are isolated polynucleotides comprising such promoters, as well as nucleic acid constructs comprising such promoters operatively linked to genes encoding a gene product, such as, a reporter, a protein, polypeptide, hormone, ribozyme, or antisense RNA, and to recombinant cells and transgenic animals comprising such nucleic acid constructs. The present invention further relates to screening methods using those recombinant cells and transgenic animals, particularly methods of screening for therapeutic compounds that modulate **tumorigenesis** and **angiogenesis**.

AN 2001:194501 USPATFULL
TI Methods and compositions for screening for **angiogenesis** modulating compounds
IN Ning, Zhang, Alameda, CA, United States
Contag, Pamela Reilly, San Jose, CA, United States
Purchio, Anthony F., Alameda, CA, United States
PI US 2001037016 A1 20011101
AI US 2000-738968 A1 20001215 (9)
RLI Continuation-in-part of Ser. No. US 1999-465978, filed on 16 Dec 1999, PENDING
DT Utility
FS APPLICATION
LREP CHARLES K. SHOLTZ, XENOGEN CORPORATION, 860 ATLANTIC AVENUE, ALAMEDA, CA, 94501
CLMN Number of Claims: 93
ECL Exemplary Claim: 1
DRWN 28 Drawing Page(s)
LN.CNT 4325

L15 ANSWER 8 OF 68 USPATFULL

AB The present invention relates to methods for inhibiting malignant tumour growth, invasion and/or metastasis in a patient, the method comprising suppressing the inhibitory activity of an inhibitor of a protease or of a non-proteolytic matrix-degrading enzyme (IPNME) in malignant tumour tissue or potential malignant tumour tissue. The suppression may be brought about by administering compounds interacting with the IPNME, but also administration of compounds interacting with transcription of genes encoding the IPNME is a possibility. The invention also relates to methods of selecting and identifying compounds in the therapeutical methods, as well of the use of such compounds in the treatment of malignancies.

AN 2001:188694 USPATFULL
TI Suppression of inhibitors
IN Brunner, Niels, Virum, Denmark
Romer, John, Copenhagen, Denmark
Ellis, Vincent, Woodford Green, Great Britain
Pyke, Charles, Copenhagen, Denmark
Grondahl-Hansen, Jan, Holte, Denmark
Pappot, Helle Pedersen, Allerod, Denmark
Hansen, Heine Hoi, Holte, Denmark
Dano, Keld, Charlottenlund, Denmark

PI US 2001034327 A1 20011025
AI US 2001-836323 A1 20010418 (9)
RLI Division of Ser. No. US 1996-583129, filed on 15 May 1996, GRANTED, Pat.
No. US 6224865 A 371 of International Ser. No. WO 1994-DK288, filed on 18 Jul 1994, UNKNOWN
PRAI DK 1993-851 19930716
DT Utility
FS APPLICATION
LREP BROWDY AND NEIMARK, P.L.L.C., 624 Ninth Street, N.W., Washington, DC, 20001
CLMN Number of Claims: 53
ECL Exemplary Claim: 1
DRWN 7 Drawing Page(s)
LN.CNT 2247
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L15 ANSWER 9 OF 68 USPATFULL

AB The invention provides isolated nucleic acid and amino acid sequences of HsKip3b, antibodies to HsKip3b, methods of screening for HsKip3b modulators using biologically active HsKip3b, and kits for screening for HsKip3b modulators.

AN 2001:163041 USPATFULL
TI Motor proteins and methods for their use
IN Beraud, Christophe, San Francisco, CA, United States
Freedman, Richard, San Mateo, CA, United States
PA Cytokinetics, Inc., South San Francisco, CA, United States (U.S. corporation)
PI US 6294371 B1 20010925
AI US 2000-621233 20000721 (9)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Prouty, Rebecca E.; Assistant Examiner: Steadman, David J.
LREP Stevens, Lauren L.Beyer Weaver & Thomas LLP
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 1645
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L15 ANSWER 10 OF 68 USPATFULL

AB The present invention relates to novel human secreted proteins and isolated nucleic acids containing the coding regions of the genes encoding such proteins. Also provided are vectors, host cells, antibodies, and recombinant methods for producing human secreted proteins. The invention further relates to diagnostic and therapeutic methods useful for diagnosing and treating diseases, disorders, and/or conditions related to these novel human secreted proteins.

AN 2001:155766 USPATFULL

TI 49 human secreted proteins

IN Moore, Paul A., Germantown, MD, United States

Ruben, Steven M., Oley, MD, United States

Olsen, Henrik S., Gaithersburg, MD, United States

Shi, Yanggu, Gaithersburg, MD, United States

Rosen, Craig A., Laytonsville, MD, United States

Florence, Kimberly A., Rockville, MD, United States

Soppet, Daniel R., Centreville, VA, United States

Lafleur, David W., Washington, DC, United States

Endress, Gregory A., Potomac, MD, United States

Ebner, Reinhard, Gaithersburg, MD, United States

Komatsoulis, George, Silver Spring, MD, United States

Duan, Roxanne D., Bethesda, MD, United States

PI US 2001021700 A1 20010913

AI US 2000-739254 A1 20001219 (9)

RLI Continuation of Ser. No. US 2000-511554, filed on 23 Feb 2000,

ABANDONED

Continuation-in-part of Ser. No. WO 1999-US19330, filed on 24 Aug 1999,

UNKNOWN

PRAI US 1998-97917 19980825 (60)

US 1998-98634 19980831 (60)

DT Utility

FS APPLICATION

LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850

CLMN Number of Claims: 23

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 15462

CAS INDEXING IS AVAILABLE FOR THIS PATENT.